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Enhanced External Counterpulsation as an Adjunct to Revascularization in Unstable Angina

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Summary: Enhanced external counterpulsation (EECP) is an effective noninvasive treatment for chronic stable angina. Despite intensive risk factor modification, a patient required two surgical coronary revascularizations and seven multivessel angioplasties over a 26-month period, demonstrating recurrent unstable angina and persistent thallium perfusion defects despite revascularization. Post EECP, angina was relieved, thallium defects were resolved, and the patient has remained asymptomatic for 36 months.

Key words: unstable angina, external counterpulsation, revascularization

Introduction

Conventional methods for treating angina include medication, percutaneous transluminal coronary angioplasty (PTCA), and coronary artery bypass grafting (CABG). Medical treatment is not always effective, and revascularization is invasive, costly, and carries a significant risk of restenosis of the affected arteries or the grafts. Repetition of these treatments is limited and may be associated with higher risks of complications.

Enhanced external counterpulsation (EECP) has been described earlier[1–5] and was modified to its present state as described in 1983.[6] Paired pneumatic cuffs are applied to the lower extremities and inflated sequentially during diastole to augment diastolic pressures and coronary blood flow (Fig. 1). EECP has been shown to be effective in the treatment of chronic stable angina.[6,7] The method was well tolerated by patients, and angina improved or stopped completely in all patients. Complete resolution of ischemic perfusion defects was achieved in 67%, partial reperfusion with decrease in size of defects was noted in 11%, and no change was seen in 22% of patients.[7] However, fixed perfusion defects did not completely resolve in any patient.

The case of a patient, with extensive triple-vessel disease and severe progressive angina despite two bypass operations and multiple angioplasties, is presented to illustrate the potential of this noninvasive procedure.

Case Report

The patient, a 58-year-old Caucasian man, had a history of mild, well-controlled hypertension, moderate obesity, low high-density lipoprotein with a normal total cholesterol, a 29-year history of smoking one and one-half packs of cigarettes a day, and of familial premature coronary heart disease. The patient was well until the beginning of April 1990 when unstable angina developed. Coronary angiography revealed severe diffuse triple-vessel disease. Because of limited graft conduits (stripping of the saphenous veins had been previously performed in 1969), a planned staged revascularization was undertaken. The left circumflex artery narrowings were dilated by balloon angioplasty and the left anterior descending and right coronary arteries were bypassed using the left (LIMA) and right (RIMA) internal mammary arteries, respectively.

Within a few months the patient developed crescendo angina, and a thallium stress study showed inferior and inferolateral ischemia. Angiography showed restenosis of the left circumflex artery, total occlusion of the LIMA graft, and severe narrowing of the RIMA. Multivessel PTCA was performed, with no marked improvement in symptoms.

In late September 1990, the angina became incapacitating with electrocardiographic (ECG) evidence of ischemia during

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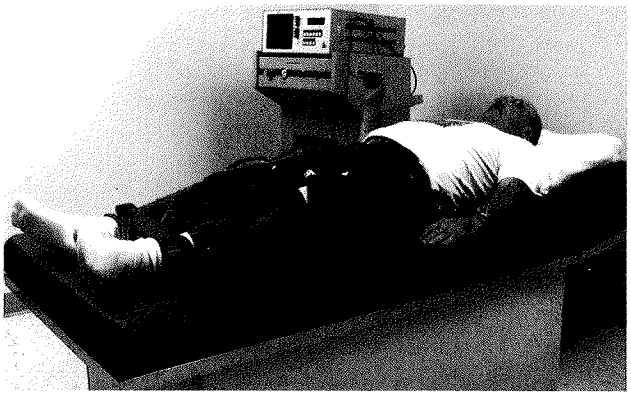

FIG. 1 Enhanced external counterpulsation device applied to patient.

pain. A second bypass operation was performed, with cephalic vein grafts placed on the left anterior descending, left circumflex, and posterior descending coronary arteries. Angiography was required for recurrent postoperative angina revealing restenosis of the left circumflex and obtuse marginal branches, severe stenoses of the cephalic vein-left anterior descending graft and RIMA-right coronary graft, and total occlusion of the LIMA and posterior descending cephalic vein grafts. Multivessel PTCA of 10 narrowings was performed with good restoration of flow.

However, recurrent angina necessitated PTCA of the first obtuse marginal branch 2 weeks later and a subsequent readmission in July 1991 for PTCA of the left circumflex and obtuse marginal branches.

Despite strict behavior modification which included a vegetarian diet, cessation of smoking, a 30 kg decrease in weight, and medication (including aspirin, Persantine®, Mevacor®), angina persisted and increased in severity, necessitating another PTCA procedure at the end of November 1991. Narrowings of the circumflex and the second obtuse marginal branches were dilated. However, within 2 weeks angina again increased in severity and a sestamibi stress test showed inferior, posterobasal, and lateral wall-fixed defects with no reperfusion. The ECG showed a right bundle-branch block and an inferolateral myocardial infarction (MI). A positron emission tomography (PET) scan with radiolabeled ammonia and F-18-FDG showed ischemia with evidence of myocardial viability in the areas considered infarcted by scintigraphy and ECG.

To save these extensive jeopardized areas, an angioplasty were performed. The left circumflex and second obtuse marginal branches were successfully dilated. A first marginal branch was totally occluded proximally with retrograde collateral flow. The persistently occluded first obtuse marginal branch was felt to be responsible for persistent abnormalities in the postprocedure sestamibi and PET scans. (Cardiac catheterizations and interventions are summarized in Table I.)

TABLE I Summary of cardiac catheterization and interventional results

Date	Cardiac catheterization	Intervention
4/13/90	Severe triple-vessel disease	
4/15/90		PTCA: LCx
4/17/90		CABG: LIMA-LAD, RIMA-RC
7/15/90	100% occulsion LIMA-LAD	PTCA: LCx, RIMA-RC
10/2/90		C ABG: CVG-LAD, CVG-OM2, CVG-RPDA
2/27/91	100% occulsions: LAD, RC, CVG-OM2, CVG-RPDA	
3/6/91		PTCA: CVG-LAD, LAD, LCx, OM1, OM2, RIMA-RC
3/25/91		PTCA: OM1
7/5/91		PTCA: CVG-LAD, LCx, OM1
10/29/91		PTCA: LCx, OM2
1/29/92	100% occulsion OM1	PTCA: LCx, OM2

Abbreviations: PTCA = percutaneous transluminal coronary angioplasty, CABG = coronary artery bypass grafting, LCx = left circumflex, OM1 = first obtuse marginal, OM2 = second obtuse marginal, LAD = left anterior descending, RC = right coronary, RPDA = right posterior descending artery, LIMA = left internal mammary artery graft, RIMA = right internal mammary artery graft, CVG = cephalic vein graft.

Methods

EECP treatment began 2 weeks after the 1/29/92 PTCA. Pneumatic cuffs were placed over the calves, thighs, and upper thighs, and timed sequential diastolic inflation of the cuffs provided augmentation of the diastolic blood pressure and coronary blood flow. An aggressive protocol was used employing higher than usual pneumatic pressures and "tighter" ECG synchronization. Under this protocol, over a period of several weeks, a dramatic improvement started to take place: the patient could gradually discontinue using sublingual nitroglycerin and Nitroderm® patches, and walking capacity improved significantly.

A thallium stress test was performed after 120 h of EECP showing total resolution of the previously noted ischemic and fixed defects. The patient has presently remained asymptomatic for more than 36 months after the last angioplasty.

Discussion

It was shown that the physiologic effects of external counterpulsation are achieved by increasing myocardial oxygen supply through increasing diastolic pressure and by reducing cardiac workload by reducing left ventricular afterload. The clinical efficacy of counterpulsation probably is related to the

degree of diastolic augmentation, that is, to the rise of the diastolic pressure to levels equal to or exceeding the systolic pressure. Zheng *et al.* studied the effects of EECP in a dog model of acute MI.[6] They showed that the mean peripheral pressure of the coronary arteries was proportional to the diastolic augmentation. Furthermore, collateral circulation was improved in dogs submitted to diastolic augmentation of such degree that diastolic pressure approached or exceeded systolic pressure, while no collaterals formed in dogs treated with nonsequential counterpulsation, which did not result in diastolic augmentation.

The accumulated evidence seems to indicate that best results are achieved by (1) using sequenced counterpulsation, that is, pressure is applied to the calves, then the lower thighs, and finally the upper thighs; (2) maximizing the volume of blood being moved during pumping—this being achieved by applying pressure cuffs over the calves, thighs, as well as over the buttocks. The inclusion of the upper limbs was not found to be essential; and (3) maximizing the pressure applied to the limbs during diastole and by proper time-sequencing of the pressure synchronized to the QRS complex.

The case described is characterized by repeated and rapid restenoses of grafts and native coronaries, necessitating two bypass operations and numerous dilatations by PTCA. The severity of the coronary pathology was of such degree that so-called fixed perfusion defects resulted which did not fill in even after thallium-201 (^{201}Tl) injections at rest. The PET study, however, indicated that the tissue was still viable and that efforts to save that jeopardized area were warranted. Initial repeat ^{201}Tl and PET studies a few weeks after the last PTCA were unchanged. However, after EECP of approximately 120 h, a turning point became apparent as the angina decreased and ceased and the patient could discontinue using nitroglycerin. The effort tolerance increased and a repeat ^{201}Tl study became normal.

Although anginal symptoms may lessen or even disappear spontaneously, this is probably the first description of a case of persistent fixed perfusion defects resolving without PTCA or surgical intervention. Treatment in this case was also associated with stabilization of what had become a virulent biologic process.

It is probable that normalization of the perfusion scan was caused by EECP treatment promoting the development or opening of collateral channels. Proof of this hypothesis would have been possible by angiography and distal occluded pressures, but the procedure was not performed in this patient given the numerous angiography and PTCA procedures and impaired renal function. In view of the follow-up of this patient, the results described in the published series, as well as the experimental data, it appears very likely that EECP is indeed promoting collateral development; however, collateral development alone may be inadequate to explain this patient's clinical course.

This is the first reported case of EECP used as an adjunct to angioplasty for unstable angina. It is possible that EECP may have normalized coronary endovascular function, relieving anginal symptoms and stabilizing the aggressive atherosclerotic and fibrointimal hyperplastic processes manifest in this patient. It is apparent from this case that EECP is a promising modality for the treatment of unstable angina in conjunction with angioplasty.

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